CASE REPORT

Latent Atriofascicular Pathway Participating in a Wide Complex Tachycardia: Differentiation from Ventricular Tachycardia

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Accessory pathways with anterograde decremental conduction properties usually are characterized by presence of antegrade preexcitation during atrial pacing. We report a 38-year-old man with frequent episodes of palpitation. No evidence of ventricular preexcitation was seen during sinus rhythm or atrial pacing. All electrophysiologic maneuvers were compatible with an antidromic tachycardia using atriofascicular pathway as the antegrade limb and the atrioventricular nodal pathway as retrograde limb. Radiofrequency ablation at recording site of accessory pathway potential resulted in cure of tachycardia with no recurrence during 3-month follow-up. This report indicated that atriofascicular pathway-mediated tachycardia should be considered in differential diagnosis of all cases of wide complex tachycardia with left bundle branch morphology and left axis. (PACE 2006; 29:1434–1437)

latent atriofascicular pathway*, *wide complex tachycardia*, *electrophysiology

Introduction

In a series of papers published between 1932 and 1947, Mahaim and his coworkers originally described anatomical connections of the atrioventricular (AV) node to the bundle branches or ventricular myocardium, bypassing the His bundle, in pathological specimens.[1–3] The true nature of these pathways was first characterized by Klein et al.[4] and Tchou et al.[5] at almost the same time. These fibers were later thought to be cause of an unusual form of preexcitation syndrome which was characterized by a left bundle branch block morphology (LBBB) tachycardia with long AV and short ventriculoatrial (VA) intervals.[6] Currently, it is demonstrated that these pathways located on the right atrium (crossing the tricuspid annulus on the free wall rather than the septum). There are several variants for these fibers; fibers that mediate tachycardia with true nodofascicular connection,[7] left-sided decremental atrioventricular connection[8] and concealed nodoventricular fiber[9] that conducting only in retrograde direction. There is only one report of latent atriofascicular pathway (evident only during antidromic reciprocating tachycardia with no anterograde conduction during sinus rhythm or atrial pacing) in the literature.[10] Here we present another interesting case of latent atriofascicular pathway who followed for many years as a case of ventricular tachycardia.

Case Report

A 38-year-old man with no evidence of structural heart disease referred to our center for evaluation of long-standing palpitation. The structural heart disease was excluded by physical examination and transthoracic echocardiography. Transthoracic echocardiography showed normal cardiac chambers (including right ventricle), normal valvular function, and ejection fraction without any wall motion abnormalities. During an episode of palpitation, the standard 12-lead electrocardiogram (ECG) showed documented wide complex tachycardia with a heart rate of 210 beats per minute (bpm). The tachycardia was refractory to two antiarrhythmics (verapamil, sotalol). The wide complex tachycardia had LBBB morphology and left axis (Fig. 1). The baseline ECG showed no abnormality.

After obtaining written informed consent, electrophysiologic study was done in the postabsorptive and nonsedated state. All antiarrhythmic drugs were interrupted for at least five half-lives before procedure. At electrophysiology study, rapid pacing from high right atrium (HRA) (Fig. 2A), different parts of right atrium (RA) and coronary sinus ostium failed to demonstrate

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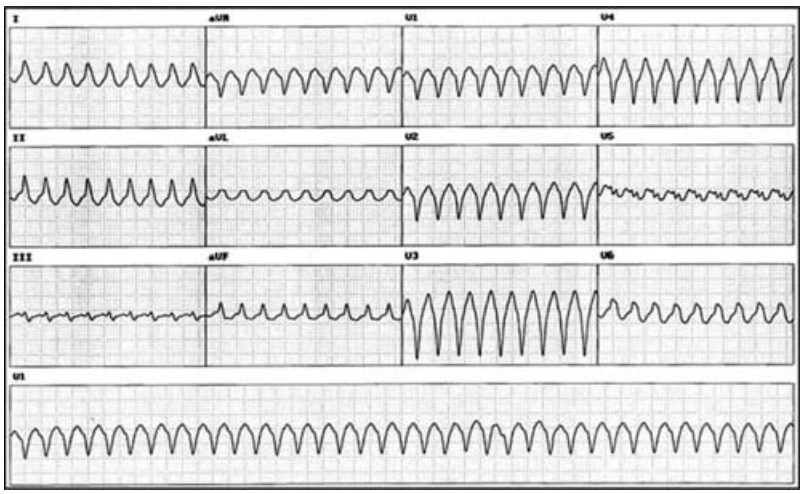

Figure 1. *Twelve-lead ECG obtained during palpitation showing wide complex tachycardia with LBBB morphology and left axis.*

preexcitation with and without isoproterenol. Dual AV nodal physiology was evident by atrial-His (AH) jump without reentry. During pacing from right ventricular apex (RVA), retrograde conduction occurred via AV node. Atrial stimulation failed to induced arrhythmia but ventricular extrastimulation (S1 = 400, S2 = 270 ms) resulted in the induction of a wide QRS tachycardia (Fig. 2B) with the following characteristics: cycle length = 280 ms; LBBB morphology and left axis; His-ventricular (HV) shortening (from 50 ms to −16 ms). During tachycardia, sequence of His bundle activation was reversed consistent with retrograde activation of this bundle (Fig. 2B). Tachycardia could be entrained from lateral RA and terminated by both RA and RVA pacing. Delivery of atrial extrastimuli from lateral RA advanced subsequent ventricular activation without affecting the timing of the atrial signal in His bundle recording (Fig. 2C). During atrial fibrillation (AF) rhythm that occurred during atrial pacing, some conducted beats characterized by LBBB and left axis morphology identical to that observed in tachycardia, HV shortening, and reversal of His bundle activation (Fig. 2D).

By impression of latent atriofascicular pathway, tricuspid annulus was mapped during tachycardia. During mapping of lateral tricuspid annulus (9 o'clock), catheter bump resulted in tachycardia termination at the site of discrete potentials. Radiofrequency (RF) energy application at this site resulted in accelerated preexcited rhythm and then loss of preexcitation and noninducibility of tachyarrhythmia. During 3-month follow-up, he was free of symptoms with no antiarrhythmic drugs and no recurrence of tachycardia was seen.

Discussion

In this report, we describe a case of wide complex tachycardia compatible with antidromic reciprocating tachycardia using a right-sided atriofascicular pathway as the antegrade limb and the AV node as the retrograde limb. The distal insertion was considered fascicular (atriofascicular fiber) because the earliest ventricular activation was recorded in the right ventricular (RV) apical region rather than tricuspid valve ring and Ventricular-His (VH) interval is relatively short (16 ms). The unusual feature in this case was the absence of anterograde conduction during sinus rhythm and pacing from several right atrial and coronary sinus sites. The anterograde conduction through atriofascicular pathway was evident only during antidromic reciprocating tachycardia and intermittently during atrial fibrillation.

The amount of preexcitation is determined by the competition between conduction through AV node and accessory pathway. Therefore lack of preexcitation during atrial pacing or sinus rhythm can be explained by combination of the accelerated AV nodal conduction and/or prolonged conduction through the accessory pathway. Another explanation for it can be concealed retrograde penetration of accessory pathway by conducted impulse through AV node.[10] In our case, short AH interval during sinus rhythm and atrial pacing and long atrium to accessory pathway potential during antidromic reciprocating tachycardia are in favor of former mechanism.

In our patient, other evidence for conduction through atriofascicular pathway was runs of overt preexcitation followed by runs of absent preexcitation during atrial fibrillation (Fig 2D). It appears that concealed conduction is the most

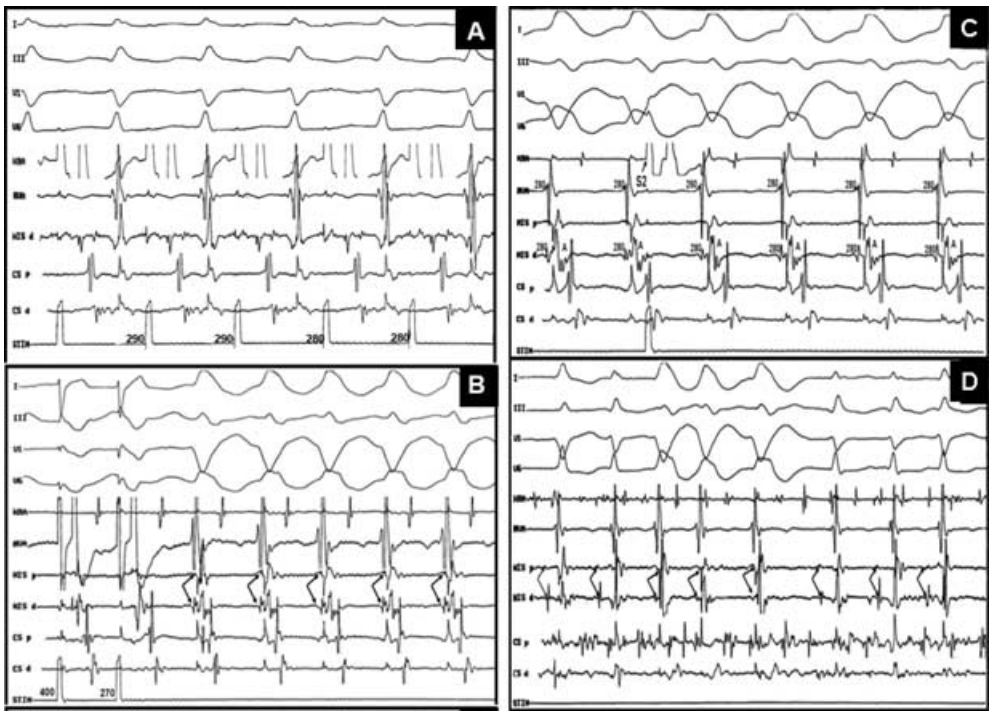

Figure 2. *(A) Part of incremental right atrial pacing at faster rates approaching tachycardia cycle length (290 and 280 ms) where AV node conduction would be slower. Note that there is no evidence of anterograde conduction through atriofascicular pathway. (B) Onset of clinical tachycardia by programmed ventricular stimulation. Tachycardia characterized by left bundle branch morphology, VH interval of 16 ms and reversal of His bundle activation. The arrows indicate retrograde His bundle activation from distal to proximal during atriofascicular pathway conduction. (C) Antidromic reciprocating tachycardia is reset (20 ms) with a late atrial extrastimulus (S2) delivered from the lateral right atrium without anterograde penetration into the normal AV node and without a change in the QRS morphology, ventricular activation sequence and timing indicating the extranodal origin of atriofascicular pathway and excluding ventricular tachycardia. (D) During atrial fibrillation, runs of overt preexcitation followed by runs of absent preexcitation. Some conducted beats (three beats in the middle of tracing) characterized by left bundle branch morphology identical to that observed in tachycardia, HV shortening, and reversal of His bundle activation. In first two beats and last three beats, arrows indicate anterograde His bundle activation from proximal to distal during AV nodal conduction. The arrows in the middle of tracing indicate retrograde His bundle activation from distal to proximal during atriofascicular pathway conduction. HRA = recording from distal poles of HRA catheter; RVA = recording from distal poles of RVA catheter; His-p = recording from proximal poles of His catheter; His-d = recording from distal poles of His catheter; CS = recordings from proximal and distal poles of multipolar coronary sinus catheter; A = atrial activation; I, III, V1, and V6 = surface ECG leads.*

possible explanation for this phenomenon. This observation is another evidence for importance of accelerated AV nodal conduction in preventing expression of antegrade conduction through atriofascicular pathway.

Another interesting feature in our patient was the presence of single wide complex beat having the identical morphology to that of preexcited beat with no preceding P-wave (Fig. 3). We considered two possibilities for this feature: (1) two-for-one phenomenon; (2) premature ventricular beat. The QRS morphology identical to that observed during antidromic reciprocating tachycardia, relatively short His-atrial (HA) interval in His electrogram and its disappearance after ablation are all in favor of the first possibility. The two-for-one phenomenon was the only evidence for presence of anterograde conduction through atriofascicular pathway during sinus rhythm. In this situation, one sinus beat was conducted down both fast AV nodal pathway and slow AV nodal and atriofascicular pathways, in which case atriofascicular pathway acted as innocent bystander. This cooperation between dual AV nodal physiology

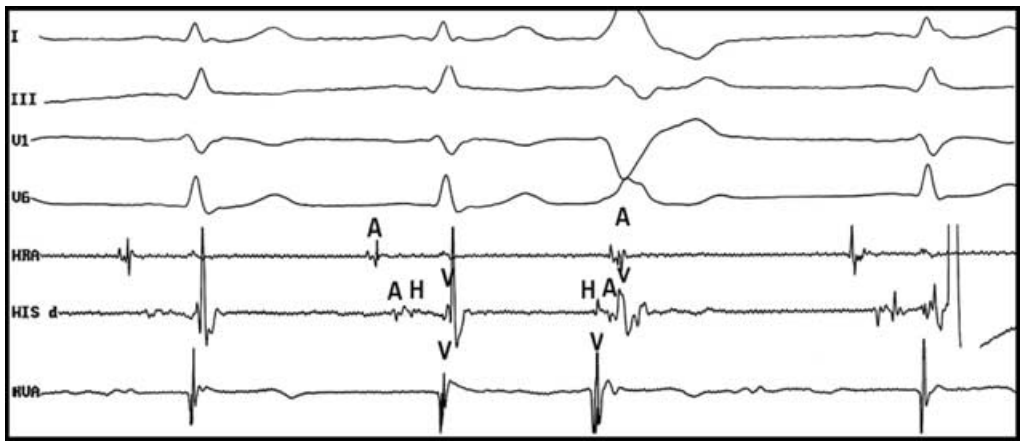

Figure 3. *2-for-1 phenomenon observed during sinus rhythm, in which an spontaneous sinus impulse produces two QRS complexes, first beat narrow complex and second wide complex, presumably resulting from conduction down both fast AV nodal pathway (narrow beat) and simultaneous slow AV nodal and atriofascicular pathways (preexcited beat). This was the only evidence for anterograde conduction seen during sinus rhythm.* ***A*** *= atrial activation;* ***H*** *= His bundle activation;* ***V*** *= ventricular activation; HRA = recording from distal poles of HRA catheter; RVA = recording from distal poles of RVA catheter; His-d = recording from distal poles of His catheter; I, III, V1, and V6 = surface ECG leads.*

and atriofascicular pathway only resulted in a single echo beat without induction of AV nodal reentry. No such triple conduction occurred after successful catheter ablation of the atriofascicular pathway.

There is only one prior report of "latent atriofascicular pathway."[10] However, there are a previous report of latent accessory pathways with decremental properties in other locations, one in the right posteroseptal area and the other in the left posterior region.[11] In the first cases of latent atriofascicular pathway, Davidson et al.[10] reported many observations like those in our case: longstanding history of wide complex tachycardia; lack of preexcitation in baseline 12-lead ECG; arrhythmia induction only by the programmed ventricular stimulation; intermittent ventricular preexcitation during atrial fibrillation; and long-term cure of arrhythmia by delivery of RF energy at recording site of accessory pathway potential. As we mentioned earlier, the initial diagnosis had been ventricular tachycardia in the case described here, and Davidson et al. were faced with the same dilemma. We excluded this diagnosis by ability of the late atrial extrastimuli delivered during tachycardia to advance ventricular activation without entering AV node, indicating engagement of atriofascicular pathway in the tachycardia circuit.

This case report reinforces importance of the diagnostic maneuvers (continuous and single atrial extrastimulation) in every case of the wide complex tachycardia with LBBB morphology and left axis despite absence of antegrade preexcitation during atrial pacing.

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